

Table of Contents (continued)T
O
R
A
C
I
A
LC
H
DC
S
PG
T
SA
C
D**1051 Regional low-flow perfusion improves neurologic outcome compared with deep hypothermic circulatory arrest in neonatal piglets***Richard J. Myung, MD, Matus Petko, MD, Alexander R. Judkins, MD, Gregory Schears, MD, Richard F. Ittenbach, PhD, Robert J. Waibel, BS, and William M. DeCampi, MD, PhD, Philadelphia, Pa*

Regional low-flow perfusion, used as an alternative to deep hypothermic circulatory arrest during congenital heart surgery, decreases neuronal injury and improves early postoperative neurologic recovery in a chronic neonatal piglet model.

1058 Inhaled prostacyclin is safe, effective, and affordable in patients with pulmonary hypertension, right heart dysfunction, and refractory hypoxemia after cardiothoracic surgery*Charl J. De Wet, MBChB, David G. Affleck, MD, Eric Jacobsohn, MBChB, MHPE, FRCPC, Michael S. Avidan, MBBCh, Heidi Tymkew, MHS, Laureen L. Hill, MD, Paul B. Zanaboni, MD, PhD, Nader Moazami, MD, and Jennifer R. Smith, PharmD, St Louis, Mo*

We prospectively enrolled 126 cardiothoracic surgical patients with pulmonary hypertension, right ventricular dysfunction, or refractory hypoxemia to receive inhaled prostacyclin. This therapy was shown to be safe and effective. Compared with inhaled nitric oxide, the calculated potential cost savings were significant.

1068 The Coapsys device to treat functional mitral regurgitation: In vivo long-term canine study*Masahiro Inoue, MD, PhD, Patrick M. McCarthy, MD, Zoran B. Popović, MD, Kazuyoshi Doi, MD, Soren Schenk, MD, Hassan Nemeh, MD, Yoshio Ootaki, MD, PhD, Michael W. Kopcak, Jr, BA, Raymond Dessoffy, AA, James D. Thomas, MD, and Kiyotaka Fukamachi, MD, PhD, Cleveland, Ohio*

The Coapsys device (Myocor, Inc, Maple Grove, Minn) significantly decreased mitral regurgitation in a canine pacing-induced functional mitral regurgitation model and maintained the results chronically.

General Thoracic Surgery (GTS)**1078 Combined proteasome and histone deacetylase inhibition in non-small cell lung cancer***Chadrick E. Denlinger, MD, Michael D. Keller, BS, Marty W. Mayo, PhD, R. Michael Broad, PhD, and David R. Jones, MD, Charlottesville, Va*

Non-small cell lung cancer appears to be resistant to histone deacetylase inhibitors in part because of activation of the antiapoptotic transcription factor nuclear factor κ B. Combined proteasome and histone deacetylase inhibition markedly enhances apoptotic cell death in non-small cell lung cancer. Future investigations with this novel combination are warranted.